The correspondence section is a public forum and, as such, is not peer-reviewed. EHP *is not responsible for the accuracy, currency, or reliability of personal opinion expressed herein; it is the sole responsibility of the authors.* EHP *neither endorses nor disputes their published commentary.*

Dimethylamine Borane Neurotoxicity

In a recent article, Tsan et al. (2005) reported the first study of the clinical toxicity of dimethylamine borane (DMAB) poisoning, particularly the neurologic manifestations in the central nervous system and peripheral nervous system. They decribed a prominent cerebellar dysfunction including impairments in performing tandem gait, finger-to-nose, and heel-to-knee tests and slurred speech in one patient who had the most severe neurologic symptoms 3 days after DMAB intoxication. We had the opportunity to evaluate the same patient 1 month after DMAB intoxication (Kuo et al., in press). Neurologic examinations revealed no cerebellar signs. Although Tsan et al. (2005) reported that brain magnetic resonance images (MRI) showed a symmetric increase in signal intensity at the bilateral cerebellar periventricular areas on 9 February 2004, the follow-up brain MRI study in our hospital 6 weeks after intoxication showed a nearly normal result. However, Tsan et al. (2005) interpreted that the rapid recovery of brain MRI changes may be due to a transient demyelination, axonal degeneration, or neuronal damages. This phenomenon is best interpreted as a transient brain edema. In addition to the transient edema, the other possibility is that the high signal intensity in cerebellar dentate nuclei is caused by an artifact. Therefore, a series section of the brain MRI images is required.

Six weeks after DMAB exposure, we found that the patient was still confused about time (Kuo et al., in press). Fourteen weeks after exposure, a neuropsychological study revealed that he had a cognitive impairment in learning ability of verbal and nonverbal learning memory; attention functions including focus, tracking, and divided attention; working memory; and semantic category retrieval. The data suggested that a prolonged toxic effect of the DMAB on the central nervous system still persisted in the attention, personality, and memory function.

The patient developed a progressive distal numbness and limb weakness 5 days after DMAB intoxication, and neurologic examinations showed generalized hyporeflexia; decreased sensation in pin-prick, temperature, touch, vibration, and position sensations; and weakness in the distal limbs, particularly in both feet (Kuo et al., in press). The data were further supported by sural nerve biopsy and cutaneous nerve biopsy studies (Kuo et al. in press). Our serial nerve conduction studies also confirmed axonal polyneuropathy, particularly in the motor nerves. However, Tsan et al. (2005) stated that the polyneuropathy with axonal polyneuropathy was verified by serial electroencephalogram and nerve conduction velocity studies.

Our time course of DMAB-induced polyneuropathy indicated an acute axonal polyneuropathy (Kuo et al., in press). However, Tsan et al. (2005) misdiagnosed the patient as having a delayed polyneuropathy, which may be wrong, because the time course was different from the so-called "delayed polyneuropathy" induced by organophosphate intoxication (Senanyake and Karalliedde 1987).

The authors declare they have no competing financial interests.

Chin-Chang Huang
Hung-Chou Kuo
Chang Gung Memorial Hospital
Taipei, Taiwan
E-mail: cch0537@adm.cgmh.org.tw

References

Kuo HC, Huang CC, Chu CC, Chu NS. In press. Axonal polyneuropathy after acute dimethylamine borane intoxication. Arch Neurol.

Senanayake N, Karalliedde L. 1987. Neurotoxic effects of organophosphorus insecticides. N Engl J Med 316:761–763.

Tsan YT, Peng KY, Hung DZ, Hu WH, Yang DY. 2005. Case report: the clinical toxicity of dimethylamine borane. Environ Health Perspect 113:1784–1786.

Dimethylamine Borane: Tsan and Hung Respond

The magnetic resonance imaging (MRI) studies of our patient (Tsan et al. 2005) were performed on the 8th and 37th days after DMAB poisoning and show marked differences. The lesions of the bilateral cerebellar periventricular area were revealed in every mode of images (fluid-attenuated inversion recovery, T2-weighted intensity, diffusion-weighted images, and T1WI) and were comparable with the patient's symptoms and signs. The possibility of artifact is small. The changes in the serial MRI suggest a transient brain lesion, which may be due to transient demyelination, neuronal damage, or edema.

We appreciate that Kuo et al. (in press) performed the nerve biopsy to prove the axonal polyneuropathy. Their result was comparable with those of our study: DMAB intoxication can lead to acute cortical, cerebellar lesions and polyneuropathy.

The authors declare they have no competing financial interests.

Yu-Tse Tsan
Dong-Zong Hung
Taichung Veterans General Hospital
Taichung, Taiwan
E-mail: hdz66@vghtc.gov.tw

References

Kuo HC, Huang CC, Chu CC, Chu NS. In press. Axonal polyneuropathy after acute dimethylamine borane intoxication. Arch Neurol.

Tsan YT, Peng KY, Hung DZ, Hu WH, Yang DY. 2005. Case report: the clinical toxicity of dimethylamine borane. Environ Health Perspect 113:1784–1786.

Sources of Lead in Cocoa and Chocolate

The article "Lead Contamination in Cocoa and Cocoa Products: Isotopic Evidence of Global Contamination" (Rankin et al. 2005) has attracted international attention (crienglish.com 2005) because of an interview that Rankin granted a reporter from *Science News* (Raloff 2005). Raloff's report makes it generally known that Rankin et al.'s study was commissioned by the American Environmental Safety Institute (AESI).

I feel that the study by Rankin et al. contains careless and misleading science, specifically Figures 2 and 4, which purport to display the isotopic measurements they listed in Table 3. However, there appear to be twice as many symbols representing chocolate products in each of these figures as there are analyses in Table 3. On closer examination, it seems that the data are shown twice: first as given in Table 3, and then a second time with each data point shifted to the right and slightly down. Furthermore, in both figures "Cocoa powder 2" (Table 2) is incorrectly plotted. If the second set of points is omitted and the "Cocoa powder 2" is replotted, a different picture emerges, one in which the chocolate products and the cocoa define two trends that lie on either side of the trend of the aerosols. Thus, the statement by Rankin et al. (2005) that "the plot shows that isotopic compositions of all the chocolate products overlap with those of lead aerosols" is unsupported by their primary measurements, and all ensuing arguments pertaining to the contamination of chocolate products during manufacture are invalid.

There are other statements in their article that I consider irresponsible. For example, Rankin et al. (2005) stated that the presence of contaminant lead in cocoa bean shells is substantiated by the concentrations of lead in the soil profiles. Such a view ignores that lead is a naturally occurring element and that the

lead content of a soil depends on how much the parent material contained and the manner in which the soil developed. In the United States, agricultural soils with the lowest lead contents are leached ultisols containing 8 ppm and those with the highest are the clay-rich vertisols with 17 ppm (Helmke 2000, Table 1.4). Seen in this light, the value of 14.2 ppm for the average of the Nigerian samples suggests minimally contaminated soil. If Rankin et al. (2005) had included the isotope ratios of the aerosols in their Figure 3, it would have been obvious that the soil leads occupied a different field. In Figure 2, for example, there are no aerosol leads with $^{208}Pb/^{207}Pb$ ratios > 2.48, whereas in Figure 3, 14 of 18 soil samples have ratios greater or equal to this value. The variability of the isotope ratios of the soils does not reflect the multiple sources of contamination that Rankin et al. (2005) invoked but rather the variable ratios of thorium to uranium in the old granitic rocks from which the soils were derived.

Another criticism I have is that Rankin et al. (2005) blurred the facts as they proceeded with their arguments. In "Sample collection and preparation" and in the title of Table 1, they pointed out that two types of beans and shells were analyzed, those taken directly from the husk (by which they presumably mean the pod) and those that had been fermented and dried. In Table 4, however, the distinction between the two types of shell was unspecified and they are listed as "Shell 1" and "Shell 2." Thereafter, both shells are treated as the same entity, with high lead contents inferred to result from atmospheric contamination. But the shells of the beans taken directly from the pod should never have been exposed to atmospheric deposition. Why then do they have lead concentrations and isotope ratios similar to those that were fermented? The simplest explanation is that both were accidentally contaminated after they were collected.

Finally, having been involved in the case of AESI *v.* Mars, Inc. et al. (AESI 2002), I know that two of Rankin's coauthors were expert witnesses for the plaintiffs and that at least one of them was compensated. I am surprised that they declared that they had no competing interests.

The author was a paid expert witness for candy companies named in a lawsuit. He currently has no consulting agreement with the defendants and was not paid or offered any form of compensation for writing this letter.

William I. Manton
University of Texas, Dallas
Richardson, Texas
E-mail: manton@udallas.edu

References

AESI. 2002. American Environmental Safety Institute *v.* Mars, Inc., Nestle USA, Inc., Kraft Foods North America, Inc., Rocky Mountain Chocolate Factory, Inc., and See's Candies, Inc. Case No. BC273433 Superior Court of the State of California for the County of Los Angeles, Los Angeles, CA. Available: http://www.aesi.ws/documents/Aesi%20v.%20Mars%20Complaint.pdf [accessed 22 January 2006].

crienglish.com. 2005. Chocolate more lead-contaminated foods. Available: http://en1.chinabroadcast.cn/2239/2005-11-6/65@280451.htm [accessed 22 January 2006].

Helmke PA. 2000. Soil Chemistry. In: Handbook of Soil Science (Sumner ME, ed). Boca Raton, FL:CRC Press, B3–B24.

Raloff J. 2005. Leaden Chocolates. Sci News 168(19). Available: http://www.sciencenews.org/articles/20051105/food.asp [accessed 22 January 2006].

Rankin CW, Nriagu JO, Aggarwal JK, Arowolo TA, Adebayo K, Flegal AR. 2005. Lead contamination in cocoa and cocoa products: isotopic evidence of global contamination. Environ Health Perspect 113:1344–1348.

Lead in Cocoa and Chocolate: Rankin and Flegal Respond

We thank Manton for his comments on our study (Rankin et al. 2005), and we hope that readers will be encouraged to read the entire article to place his correspondence in perspective. In our article (Rankin et al. 2005), we documented the orders of magnitude of increases in lead concentrations of processed cocoa and chocolate products over that of the lead concentration in cocoa bean nibs used in the manufacture of those products. We maintain that our conclusions are substantiated by both the data and the extensive literature documenting industrial lead contamination in the biosphere (Cocoa Producers' Alliance 2004; National Research Council 1993]. Our lead concentration measurements show that manufactured cocoa and chocolate products exhibit contamination not found in the source material—cocoa bean nibs. As detailed in the article, the average lead concentration of the cocoa bean nibs was among the lowest reported values for a natural food, whereas the lead concentrations of the cocoa products were up to over 300-fold higher; as we noted in our article (Rankin et al. 2005), those relatively high levels of lead have been acknowledged by the Cocoa Producers' Alliance (Lagos, Nigeria).

Because the concentration measurements indicated that this contamination was not naturally derived, we analyzed other materials for lead concentrations and lead isotopic composition to determine possible sources of the increase in lead in cocoa products. We are unsure about the origination of additional points in Figures 2 and 4, but we used the data listed in Table 3 in forming our original conclusions, and we continue to support those conclusions. To suggest, as Manton does, that the lead isotopic composition measurements show that the contamination is naturally derived is disingenuous and inconsistent with the extensive literature using lead isotopic compositions to characterize potential sources—natural and industrial—of lead in the biosphere (Cocoa Producers' Alliance 2004; National Research Council 1993). We maintain that the lead isotopic composition measurements suggest that, although it may be possible for some contamination originating at the cocoa farms to be transferred to the final products, the majority of contamination must accumulate during shipping and/or manufacturing, as both the lead concentrations and lead isotopic compositions of cocoa and chocolate products indicate.

Finally, we are disappointed with Manton's comment regarding our declaration of no competing financial interests. In our article (Rankin et al. 2005), we listed the American Environmental Safety Institute (AESI) as a source of funding. The institute provided some of the funding for the chemical analyses, scientific interpretation, and litigation depositions, but these were all done with the agreement that our study would be equipoised—conducted without bias. Moreover, the litigation was settled well before our manuscript was written, and we neither received nor requested funding from the AESI or any other organization or any individual involved in the litigation for the preparation or publication of the article.

The authors declare they have no competing financial interests.

Charley W. Rankin
A. Russell Flegal
Environmental Toxicology, WIGS,
University of California, Santa Cruz
Santa Cruz, California
E-mail: rankin@etox.ucsc.edu

References

Cocoa Producers' Alliance. 2004. Proposed Methodology to Determine Source and Level of Lead Contamination in Cocoa. Available: http://www.copal-cpa.org/lead.html [accessed 26 January 2005].

National Research Council. 1993. Measuring Lead Exposure in Infants, Children and Other Sensitive Populations. Washington, DC:National Academy Press.

Rankin CW, Nriagu JO, Aggarwal JK, Arowolo TA, Adebayo K, Flegal AR. 2005. Lead contamination in cocoa and cocoa products: isotopic evidence of global contamination. Environ Health Perspect 113:1344–1348.